Surgical management of congenital extrahepatic portosystemic shunts in dogs and cats

Chirurgische behandeling van congenitale extrahepatische portosystemische shunts bij honden en katten

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ABSTRACT

A portosystemic shunt is an erroneous venous communication between the portal vein and the systemic circulation. Surgery is the treatment of choice. Progressive attenuation of the shunt is indicated in most cases to prevent portal hypertension. Gauged attenuation using silk was the first method used. Different methods are used to estimate the degree of closure that can be tolerated by the patient and will not cause portal hypertension, but they are time-consuming and not always equally reliable. If only partial closure is possible, a second surgery may be needed. Gradual attenuation can be achieved using an ameroid constrictor (AC), cellophane banding (CB), a hydraulic occluder or thrombogenic coils. There is great interpatient variability between the rate and degree of shunt closure when using an ameroid constrictor or cellophane banding. Thrombogenic coils and hydraulic occluders appear to give a more controlled shunt closure, but further investigation is needed.

SAMENVATTING

Een portosystemische shunt is een aberrante veneuze verbinding tussen de vena porta en de systemische circulatie. Het chirurgisch ligeren van deze shunt is de beste behandeling. Bij de meeste patiënten kan enkel een geleidelijke vernauwing van de shunt bekomen worden door het ontstaan van portale hypertensie. Verschillende methoden zijn voorhanden om de mate van het afsluiten van de shunt -die door de patiënt wordt verdragen zonder dat er portale hypertensie ontstaat-, te evalueren, maar ze zijn tijdrovend en niet altijd even betrouwbaar. Het gestageerd sluiten van de shunt met behulp van een zijdeligatuur was de eerste beschreven methode. Andere methoden werden gerapporteerd, zoals de ameroïd constrictor (AC), cellofaan band (CB), trombogene coils en hydraulische occluders. De ameroïd constrictor en cellofaan band geven vaak variaties in het tijdstip waarop en de snelheid waarmee de shunt volledig sluit en ook in de mate van het afsluiten. Meer controleerbare progressieve afsluitmethoden, zoals thrombogene coils en hydraulische "occluders", worden momenteel onderzocht. Door de nieuwe technieken (AC en CB) zijn de chirurgische complicaties verminderd en de resultaten verbeterd.

INTRODUCTION

A portosystemic shunt (PSS) is an erroneous venous communication between the portal vein and the systemic circulation, which bypasses some or all of the hepatic tissue (Swalec and Smeak, 1990). A PSS can be either congenital or acquired secondary to portal hypertension. A congenital PSS consists mostly of a single vessel, but can also be multiple (with a maxi-

mum of 2 vessels), as reported in 3% and 11% of the cases, respectively, in two different studies (Johnson et al., 1987; Winkler et al., 2003). In the case of an extrahepatic PSS (EH-PSS), anomalous connections are found between the portal vein or one of its four tributaries, on the one hand, and the caudal vena cava, renal vein, azygos vein, internal thoracic vein, phrenic vein, vertebral vein or umbilical vein remnant, on the other hand (Figure 1). Sometimes there is

- 1. v. cava cranialis
- 2. v. azygos dextra
- 3. v. vertebralis
- 4. v. thoracica interna
- 5. v. intercostalis,
- 6. v. cava caudalis
- 7. vv. hepaticae
- 8. v. renalis
- 9. v. testicularis/ovarica
- 10. v. circumflexa ilium profunda
- 11. v. iliaca communis
- 12. v. iliaca interna
- 13. v. iliaca externa
- 14. vv. lumbales
- 15. v. portae: ramus sinistra(a),
- ramus dexter(b)
- 16. v. gastroduodenalis
- 17. v. lienalis
- 18. v. gastrica sinistra
- 19. v. mesenterica cranialis
- 20. v. mesenterica caudalis,
- h. heart, l. liver.

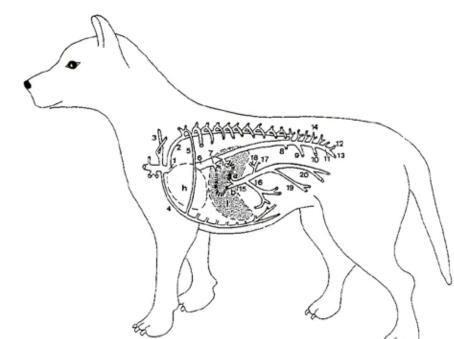


Figure 1. Compilation of the most important intrathoracic and intra-abdominal veins in the dog (left lateral view).

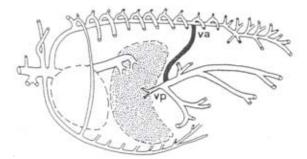


Figure 2. Anastomosis between the portal vein (vp) and the azygos vein (va), with absence of the posthepatic caudal vena cava.

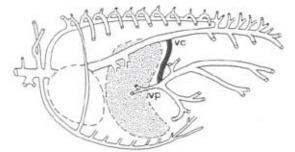


Figure 3. Direct anastomosis between the vena cava caudalis (vc) and the portal vein (vp).

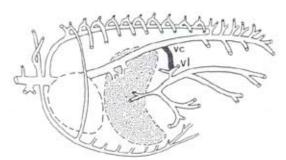


Figure 4. Congenital extrahepatic shunt between the v. lienalis (vl) and the v. cava caudalis (vc).

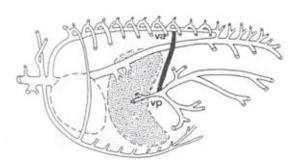


Figure 5. Congenital extrahepatic shunt between the v. porta (vp) and the v. azygos (va).

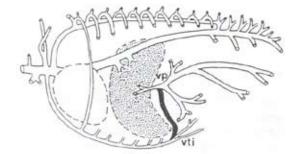


Figure 6. Congenital extrahepatic shunt between the v. porta (vp) and the v. thoracica interna (vti).

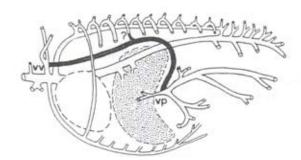


Figure 7. Congenital extrahepatic shunt between the v. porta (vp) and the v. vertebralis (vv) or the v. intervertebralis.

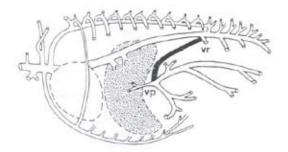


Figure 8. Congenital extrahepatic shunt between the v. porta (vp) and the v. renalis (vr).

an anastomosis between the portal vein and the azygos vein with absence of the posthepatic caudal vena cava (De Rycke *et al.* 1995) (Figure 2). EH-PSSs are primarily found in small- and toy-breed dogs such as Maltese, Yorkshire terriers, miniature schnauzers and poodles. Cats can have extrahepatic or intrahepatic shunts, although extrahepatic locations predominate (79 %) (Birchard and Scherding, 1992). Only attenuation of extrahepatic shunts will be discussed in this article.

SURGERY OF THE EXTRAHEPATIC PORTO-SYSTEMIC SHUNT

Anatomic variations and locations

The goal of surgery is to identify and occlude the shunting vessel, thereby establishing normal hepatic portal blood flow, and to do so without causing portal hypertension.

Perfect knowledge of the regional anatomy of the portal vein and caudal vena cava of the dog and cat is essential. The portal vein arises from the fusion of the cranial mesenteric and the caudal mesenteric veins and receives blood from the splenic vein and the gastroduodenal vein, on its course to the liver (Figure 1). At the liver hilus it divides into a right and a left portal vein branch in the dog, and into a left, a central and a right portal vein branch in the cat. The portal vein provides 80% of the hepatic blood flow and 50% of the oxygen supply to the liver, the rest being provided by the hepatic arteries. (Tobias and Rawlings, 1996)

The caudal vena cava is situated on the right side of the aorta and has 4 major tributaries and 4 minor tributaries. The major tributaries (from caudal to cranial) are: the common iliac veins, deep circumflex iliac veins, renal veins, and 6 to 8 hepatic veins. The minor tributaries are: the irregularly occurring lumbar veins (IV-V-VI cat), the right testicular or ovarian vein, the phrenicoabdominal vein and the phrenic vein (Barone R., 1976).

Most extrahepatic shunts are portocaval shunts (64% or 104/162 in Mehl *et al.*, 2005, 75 % or 50/66 in Kummeling *et al.*, 2004, and 80% or 110/137 in Wolschrijn *et al.*, 2000). There can be a direct anastomosis between the vena cava and the portal vein (Figure 3) or an anastomosis between one of the four tributaries of the vena porta and the vena cava. A shunt originating from the splenic vein is the most frequently encountered anatomical origin among these 4 tributaries (Figure 4).

A portoazygos shunt is seen in 20% to 24% of the cases (Wolschrijn et al., 2000; Kummeling et al., 2004), and in one study even in 36% of the cases (Mehl et al., 2005) (Figure 5). The shunting vessel can be small (3mm) and difficult to visualize when a vascular spasm induced by surgical manipulation causes it to obliterate. Most patients with a portoazygos shunt are presented at an older age (>2years) (Tobias, 2003; Mehl et al., 2005). Dachshunds and Yorkshires seem to be predisposed to portoazygos shunts. Other shunt locations are seen less frequently and may occur between the portal vein, on the one hand, and the internal thoracic vein (Figure 6), vertebral vein (Figure 7), phrenic vein, left colic vein or renal vein (Figure 8), on the other.

Surgical approach - 'finding the shunt'

The abdomen is entered very carefully through a midline celiotomy because of the possibility that the PSS may have occurred in the falciform ligament. A systematic exploration of the entire abdominal cavity is performed. As most shunts occur at the level of the epiploic foramen, between the renal and the hepatic vein, the descending part of the duodenum is retracted to the left and the epiploic foramen is inspected. In this manner, most vessels entering the right and the ventral site of the caudal vena cava can be inspected. To evaluate vessels entering the left side of the caudal vena cava, the omental bursa is opened and the duodenum and pancreas are retracted to the right side. Most portocaval shunts originating from the splenic vein or one of its tributaries can be seen through the omental bursa. Portocaval shunts are ligated as close to the vena cava as possible to ensure that no tributary is missed. The connection of the shunting vessel to the vena cava can be recognized by the turbulent flow in both vessels and the dilation of the vessel. Most shunts have an average diameter of 1 cm (ranging from 0.7-2.5 cm) (Tobias K.M. et al., 1998).

Portoazygos shunts can transverse the diaphragm. Any large vessel that enters the thorax at the level of the esophageal hiatus will be a shunt. The only vessels that may enter the aortic hiatus are the aorta, the right azygos vein and the left hemiazygos vein. Portoazygos shunts can be inspected at the level of the diaphragm by retracting the stomach caudally and the liver to the right, if the shunt enters the esophageal hiatus or the left lumbar crus. If the shunt enters the thoracic cavity at the right crus, the preferred approach is through the omental bursa by retracting the stomach cranially and the intestines caudally. Portoazygos shunts are preferably ligated at the level of the diaphragm, making sure that no tributaries (gastric or gastroepiploic vein) enter the shunt proximal to the ligation. If necessary, the diaphragm can be opened to ligate the shunting vessel intrathoracically.

After the shunt is located, the portion of the portal vein cranial to the shunt is inspected to identify agenesis or atresia of the portal vein. Over a 3-year period, K.M.S. Tobias (1998) saw only 1 dog of 39 dogs or 2.5 % with agenesis of the portal vein.

The shunt is usually tortuous and thin walled. Very careful dissection with right angled forceps is indicated, taking care never to close the forceps when dissecting behind the shunt, as this could accidental-

ly tear the vessel wall. Dissection is continued until there is no more resistance felt when passing a forceps or occluding device around the shunt.

DECIDING THE DEGREE OF VASCULAR ATTENUATION

Some EH-PSSs can be completely ligated, however in 48%-68% of the cases total ligation of the PSS cannot be achieved without portal hypertension (Lawrence *et al.*, 1992; Burton and White, 2001).

To estimate the degree of occlusion that can be achieved safely, the shunt is temporarily occluded for 5-10 minutes while the surgeon uses both subjective and objective parameters to evaluate changes.

Subjective parameters such as intraoperative evaluation of the gross appearance of the intestines and splanchnic circulation generally give a good idea of the degree of shunt attenuation that can be achieved safely (Matthews and Gofton, 1988). The first sign of an increase in venous pressure is pallor of the surface of the small intestines. Normal capillary pressure is to be maintained. An increase in venous pressure can cause a reflex increase in arteriolar resistance and a decrease in post-capillary resistance, resulting in reduction in blood flow of the capillary bed. Because of the constriction in the arterial vessels and the dilatation in the mesenteric vessels, increased mesenteric vascular pulsation can be observed. If shunt attenuation is increased and continued beyond this point (i.e. more occlusion), the hypoxia will reduce the myogenic response in the arterioles and a reactive hyperemia will cause the intestines to become cyanotic. This hypoxia can lead to increased intestinal motility. The same changes (edema and cyanosis) can be observed in the pancreas.

However, objective methods to measure the rise in portal hypertension after ligation are also described: for instance, the measurement of portal venous pressure (PVP), central venous pressure (CVP), arterial pressure (AP), heart rate (HR) and the end-expiratory CO2.

Direct measurement of the PVP can be achieved by placing a catheter into a jejunal vein or by transsplenic portal catheterization (Shultz *et al.*, 1993). When transsplenic portal catheterization is used, a large venule of the caudal pole of the spleen is selected and a catheter is inserted through the parenchyma (parietal surface of the spleen) and then advanced through the splenic vein into the portal vein. This allows the surgeon to measure the PVP directly in the portal vein. When the catheter is in place, a water manometer or a pressure transducer can be connected.

The normal PVP of a dog is between 5 and 10 mm Hg (7-14 cm H2O), which, as a result of the hepatic resistance, is 5-6 mm Hg higher than the venous pressure. In a patient with a PSS, because of the diversion of portal blood flow into the systemic circulation, the portal pressure is usually lower, ranging between 0 and 9 mm Hg (0-12 cm H2O) (Johnson *et al.*, 1987). When shunt ligation is performed, the PVP will rise and it should not be allowed to exceed 14-15.5 mm Hg or 19-21 cm H2O (Johnson, 1987; Swalec and

Smeak, 1990). The maximum change in PVP should be less than 8 mm Hg (11 cm H20) (Swalec and Smeak, 1990). If this rule is not respected, the risk for fatal portal hypertension increases substantially. PVP measurements are sensitive to variations caused by blood pressure, the anesthetic depth, the hydration status of the patient, the degree of splanchnic compliance, the amount of surgical manipulation, the phase of respiration and other systemic factors that could increase splanchnic vascular resistance and therefore lower intestinal and hepatic blood flow. Shunt attenuation based on PVP in a patient with this reduced splanchnic flow could make the surgeon decide erroneously that shunt ligation is safe. Therefore one should better rely on using both the pre- and postligation PVP. False negative estimates (i.e. 'no risk for portal hypertension' according to the measurements, while the patient died or suffered postoperatively because of portal hypertension) were reported in 6% (Lawrence et al., 1992), 8 % (Komtebedde et al., 1991) and 9 % (Swalec and Smeak, 1990) of the patients.

Normal CVP is 0 cm H2O. Increase in PVP will lead to a decrease in CVP caused by changes in venous return and splanchnic venous pooling (Swalec and Smeak, 1991). A decrease in CVP greater than 1 cm H2O has been associated with the development of postoperative portal hypertension (Swalec and Smeak, 1990).

A substantial number of articles mention the use of monitoring the changes in heart rate during shunt attenuation as a method of predicting portal hypertension. In a prospective study using 6 healthy dogs, Swalec and Smeak (1991) did not see any intraoperative changes in heart rate during temporary ligation of the portal vein. They attributed this lack of change to the effect of anesthesia. Isoflurane anesthesia may attenuate baroreceptor reflexes, and cardiac output may change without concomitant alterations in heart rate (Seagard *et al.*, 1983), although it can be helpful to monitor the heart rate in association with arterial blood pressure.

A decline in arterial blood pressure below 80 mm Hg at the carotid sinus will cause splanchnic vaso-constriction and decreased splanchnic blood flow (Brooksby G.A., Donald D.E., 1971). A significant increase in the heart rate and a decrease in the arterial blood pressure during ligation may indicate a marked decrease in venous return associated with shunt occlusion (Komtebedde J., et al. 1991).

Some patients do not tolerate any degree of ligation because of complete portal hypoplasia. Over a 3-year period, K.M.S. Tobias (1998) saw only 1 out of 39 dogs (or 2.5%) with agenesis of the portal vein.

ATTENUATION OF THE EXTRAHEPATIC PORTOSYSTEMIC SHUNT

Suture ligation

Silk ligation, which was the first method described for the ligation of PSS, is still being used. Silk is an almost non-absorbable multifilament suture. It has prolonged tissue retention, but in time disruption of the silk strands and phagocytosis of the silk by macrophages can be seen (Youmans and Hunt, 1999). Some authors mention accelerated resorption in cats and in dogs (Kummeling *et al.*, 2004). This has led to the recommendation to use polypropylene in cats.

The shunt is ligated either partially or completely, depending on the changes in the PVP and/or the physical changes occurring as a result of portal hypertension. Some authors (Van Vechten et al., 1994; Hottinger et al., 1995) state that after partial ligation, the process of shunt closure will still progress. They cite two mechanisms: the progress in inflammation and the alterations in blood flow. Surgical manipulation and the use of silk will produce an acute inflammatory reaction caused by chemotaxis and degranulation of neutrophiles. This will induce a progressive attenuation of the shunt in the acute phase. In a later phase fibroblast proliferation and scar formation will cause further attenuation. However, in an experimental study using a femoral vein in dogs over a 6 week period, Youmans and Hunt (1999) failed to confirm that partial attenuation with silk would proceed to complete attenuation. They hypothesized that this discrepancy might have been caused by a different degree of inflammation in the abdominal environment compared to the femoral region. In another study, Hunt and Hughes (1999) found that there was no significant difference in the proportion of complete occlusion when silk sutures of different gauges were used. This also would suggest that perivascular fibrosis caused by silk is not the most important factor in the gradual ongoing occlusion after the use of a silk suture. Kummeling and others (2004) hypothesized that alterations of blood flow could be the cause of the gradual venous attenuation after surgical partial ligation. When the shunt is partially ligated, the vascular resistance of the shunt increases, thus redirecting the blood flow along the path of least resistance (portal vein). Reduced portal vascular resistance due to expansion of the cranial part of the portal vein may lead to reduction of portosystemic shunting postoperatively (rather than perivascular fibrosis around the ligature). The closure of the shunt would then be caused by thrombosis secondary to stasis. Residual flow through the shunt (and therefore the final clinical outcome) depends on the ability of the portal system and the liver vasculature to adapt to pressure changes caused by narrowing of the shunt.

The current veterinary literature yields conflicting views with regards to the long-term outcome after portosystemic shunt ligation with silk. Some studies found no difference between the outcome of complete and partial closure of shunts in dogs that survived surgery (Lawrence et al., 1995; Van Vechten et al., 1994; Komtebedde et al., 1995). But others (Hottinger et al., 1995; Johnson et al., 1987; Hunt and Hughes, 1999) have reported better results after complete closure than after partial closure. Up to 41% (10, 25, 29 and 41% in the respective studies) (Kummeling et al., 2004, Smith et al., 1995; Hunt and Hughes, 1999; Hottinger et al., 1995) of the dogs with a partially occluded PSS suffer relapse of clinical signs between 18 months and 6 years following surgery. Therefore the final goal should always be to completely occlude the shunt, provided the portal pressure measurement will tolerate this course of action. However, in 48% to 68% of the dogs with a congenital EH-PSS, only partial occlusion can be achieved. If clinical signs are present and scintigraphy still indicates significant shunting 1 to 3 months after surgery, it is recommended that a second surgery be performed in an attempt to achieve complete closure of the remainder of the shunt in these patients (Hottinger *et al.*, 1995). Nevertheless, owners should be warned that in 50% of all shunts complete attenuation cannot be achieved, because of elevated portal pressure.

Long-term clinical success is described in up to 70% of the cases, when only partial attenuation was achieved in the first surgery (Hunt and Hughes, 1999). The same authors had a long-term clinical success of 92% in dogs where the shunt was completely ligated.

The advantages of silk ligation are its availability, the low cost, and the fact that it can be used in both intra- and extrahepatic shunts. The disadvantages are the need for intra-operative measurements of PVP, which leads to an increase in morbidity, and the fact that in some cases there can be a need for a second surgical procedure to attempt complete ligation since complete attenuation is not predictable. Hunt and Hughes (1999) also found that the success rate after partial ligation with silk was related to the surgeon's experience.

GRADUAL ATTENUATION

Techniques with slow ongoing shunt attenuation such as ameroid constrictors, cellophane banding, thrombogenic coils and hydraulic occluders may improve surgical outcome. Several methods for gradual vascular occlusion have been developed. As the shunting vessel is gradually occluded, the blood flow to the liver increases gradually, thus allowing the hepatic architecture to develop and thereby preventing fatal hypertension.

AMEROID CONSTRICTOR

The Ameroid constrictor (AC) is a device that was developed specifically for gradual occlusion of vessels. It has been used since 1950 in experiments to create canine models of coronary arterial stenosis. In 1996 Voght and others described the first report of gradual vascular occlusion of a PSS with the AC in multiple cases.

The constrictor has an outer ring of stainless steel and an inner ring of casein. Casein is a hygroscopic material that will expand in fluid. The outer stainless steel ring forces the casein to expand towards the centre of the ring, thus providing a gradual occlusion of the vessel. When placing the AC, minimal initial occlusion of the shunting vessel is desirable, with a maximal occlusion of 25% of the shunting vessel being allowed (Voght *et al.*, 1996) (Figure 9). To achieve this goal, ACs with internal diameters of 3.5mm, 5mm, 6mm, 6.5mm, 7mm, 7.5mm, 8mm and 9mm are available. Minimal dissection of the fascia around the PSS is done to prevent movement of the ring. The shunt is flattened by using right angled for-

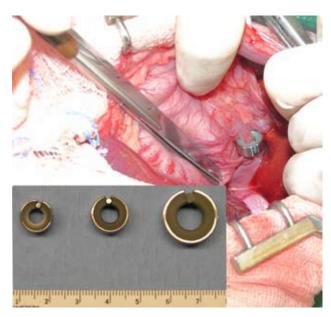


Figure 9. Intraoperative placement of an ameroid constrictor in an extrahepatic portocaval shunt at the level of the epiploïc foramen.

ceps and the AC without the key is grasped with an Allis forceps and slipped over the shunt. With a hemostat the key is placed to complete the casein ring. The constrictor is attached to a surrounding structure, thus limiting the occurrence of kinking of the vessel, which would lead to acute, premature and complete closure of the vessel.

The AC produces an early rapid occlusion in the first 2 weeks, followed by a more gradual occlusion in the following 2 months (Voght et al., 1996). However, there is great variability between the different reports of occlusion time of the AC. Voght et al. (1996) found an occlusion time of approximately 30 days in a splenic vein model in the dog. In the clinical part of the study, which involved treating 12 dogs and 2 cats, 50% of the patients developed shunt occlusion by day 30 and 22% by day 60, 90 and 120 post-AC placement, 14% died of portal hypertension Î day after surgery and 14% developed multiple extrahepatic PSS. In a study with a femoral vein model to evaluate progressive venous attenuation in the dog (Youmans and Hunt, 1999), the authors noted that in all but one instance the AC produced vascular occlusion within 7 days. Variations in the rate of vessel closure after AC application have been associated with variations in tissue reaction to the ameroid clay. In particular, the formaldehyde contained in the ameroid clay can induce substantial tissue reaction, which may predispose to thrombosis (Ikeda *et al.*, 1981). Youmans and Hunt (1999) reported the presence of a thick fibrous capsule around the AC and observed that the lumen of the AC did not close completely. Obliteration of the femoral vein in this experimental model was therefore caused by a core of dense fibrous tissue and not by the casein itself.

When surgical time is compared between ligation obtained with AC and ligation obtained with silk, 20% (Hurn and Edwards, 2003) to 50% (Murphy *et al.*, 2001) shorter ligation times are reported for AC because there is no need to measure portal pressure. Some authors have noted that the utilization of the

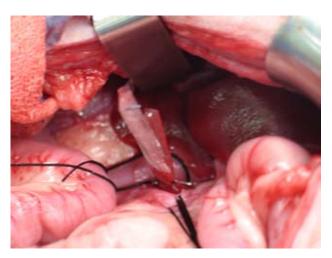


Figure 10. Intraoperative placement of a cellophane band in an extrahepatic portocaval shunt.

AC lowered the incidence of intraoperative and postoperative complications, but there was no significant statistical difference to support this finding (Murphy et al., 2001; Hurn and Edwards, 2003). Shortening the operation time is an important consideration, given that most animals are small breeds and usually small for their age. The AC may also be a good alternative in the hands of a surgeon with less experience in this domain, as it has been demonstrated that surgeon experience is a major contributing factor to the success rate when using silk ligation (Hunt and Hughes 1999).

Postoperative mortality rates of 14% (Vogt J. et al., 1996) and 7.1% (Mehl M. et al., 2005) have been reported. In a long-term follow-up of 3 years, Mehl M. et al. (2005) reported an excellent outcome in 80% of the cases. The dogs were clinically normal without medical treatment. In the same study, 14% of the owners reported a good outcome (need for diet and 2 to 3 seizures/year), and in 6% the outcome was considered poor as no improvement was seen or the dog died due to complications of PSS.

Cellophane banding

A cellophane band is made by folding a 1.2 cm wide strip of sterilized cellophane in thirds lengthwise, to form a 4 mm wide 3-layered band (Hunt et al., 2004). Cellophane of different types of origin can be used, such as household or scotch tape. Little or no research has been done on the variability of the patients' inflammatory response to cellophane of different origins. Only Youmans and Hunt (1999) mention no difference in shunt attenuation or histological tissue response in an experimental rat model between the shunt ligated with scotch tape and the other shunts ligated with non-adhesive cellophane. The cellophane band is placed around the shunt and tightened, making sure that the shunting vessel is constricted less than 50% of its internal diameter. The cellophane band is first tightened around a rod or Steinman pin with appropriate diameter using surgical clips, after which the rod or pin is removed and the ends of the cellophane band are sutured together (Figure 10). In most dogs, shunt occlusion is achieved within 8 weeks after placement of the cellophane band and

is caused by an initial acute inflammatory response followed by a chronic low grade foreign body tissue reaction. In cats, cellophane banding is not recommended because of limited inflammatory response. Additionally, Youmans and Hunt (1999) found that complete occlusion with cellophane banding was not achieved when the initial diameter of the cellophane band was more than 3 mm. They suggested that one should restrict the internal diameter of the cellophane band after ligation to 2.5mm or less, providing this does not result in portal hypertension.

The advantages of cellophane banding are the low cost, the availability and the ease of application. As for the AC, there is no need to measure the portal pressure, which shortens the operation times. The advantage of cellophane banding is that it produces a slower occlusion than an AC (8 weeks versus 2 weeks). Its disadvantages are its variability in shunt occlusion and the restriction of attenuating shunts that cannot be ligated less than 3mm without causing portal hypertension. Like silk ligation and AC placement, this method has interpatient variability caused by factors like the patient's natural degree of inflammatory response, initial degree of shunt occlusion, width of the cellophane band and type of cellophane band that is used.

Thrombogenic coils

During the last few years, developments in interventional radiology have opened up the possibility of using thrombogenic coils (TC). These coils consist of a flexible metallic strip and multiple polyester fibers to stimulate thrombosis. Under fluoroscopic control, such a coil is placed into the vessel lumen via a catheter access and guide wire. Occlusion of the vessel occurs when a thrombus develops on and around the thrombogenic materials (Partington *et al.*, 1993).

In 1993 Partington et al. published the first case report of the use of a TC in an intrahepatic shunt. In 2000 a second case report was published by Gonzalo-Orden et al. First a vena cava Wallstent (Stent Schneider, Zurich, Switzerland) was placed at the entrance of the shunt in the vena cava, to prevent migration of the TC from the shunt into the vena cava by the strong blood flow. One month later a second procedure was performed to introduce 2 thrombogenic coils in the shunting vessel. Two weeks after the second procedure, the shunt was still patent but the blood values of the dog had returned to normal and the dog was considered clinically normal. In 2003 Leveille et al., as well as Weisse et al. (2003) reported additional cases and Leveille first used the TC in EH-PSS. Both authors used a TC in combination with a Wallstent as described by Gonzalo-Orden et al. (2000). In 2007 Bussardori et al. described the treatment of 6 dogs with EH-PSS and intrahepatic PSS using transvenous coil embolization in combination with a vena cava stent. The median operation time was 58 minutes. Due to the use of vena cava stents and careful measurement of the diameter of the shunting vessel for the purposes of choosing the correct coil diameter and number of coils to be used, no coil migration was seen. In all dogs, the shunt was closed between 1 and 2 months after surgery. Upon followup, 4 dogs had no clinical signs and normal hematological values 2 years after surgery. One dog had normal values and no clinical signs, but died 6 months after surgery due to unrelated causes, and 1 dog was euthanized 6 months after partial occlusion due to hepatic insufficiency.

The advantages of the technique are the minimally invasive procedure, the short anesthetic period, the faster recovery and the shorter hospital stay. However, major disadvantages are also described, including coil migration, bleeding, the need for specialized instrumentation and training, multiple procedures and the irregular development of recanalization at the site of thrombosis.

Hydraulic occluders

None of the previously described techniques, including partial ligation with silk, AC, cellophane and TC, assures a gradual, complete occlusion. In a recent experimental study, Sereda et al. (2005) evaluated a percutaneously controlled hydraulic occluder (HO) for gradual attenuation of the vena cava in a rat. The HO consisted of an inflatable silicone membrane with a polyester-reinforced, stretch resistant cuff, a variable length of actuating tubing and a subcutaneously placed injection port. The HO was placed around a vessel and closed by placing a suture through the pre-existing holes. The HO was then connected to a subcutaneously placed injection port. Inflation of the injection port was controlled by gradual injections of physiologic saline. The occlusion was then followed weekly with the aid of a flow probe placed caudal from the HO. A gradual decrease in blood flow over 8 weeks with complete occlusion was achieved in all rats. Occlusion was caused by physical compression and not by chronic inflammation, since silicone only gives a minimal tissue reaction. These results seemed promising in the search for a reliable, gradual occlusion device that could be reversed, if necessary. The use of the HO to treat a PSS has not been reported, although the author of the previously mentioned study is currently performing a prospective clinical study in dogs with intrahepatic PSSs.

LONG-TERM OUTCOME

The long-term outcome is determined by the capability of the patient to develop an appropriate hepatic vasculature and architecture. Dogs < 12 months old at surgery can be given a better prognosis than those > 2 years old (Lawrence *et al.*, 1992). Strombeck (1977) attributed this to the ability of the liver of younger dogs to respond better to the increase in hepatotrophic factors and the less severe changes in hepatic structure.

CONCLUSION

Surgical attenuation of an extrahepatic PSS is a challenging procedure because of the need for controlled gradual vascular attenuation. Gauged attenuation using silk necessitates the measurement of PVP

or evaluation of gross appearance of the intestines and splanchnic circulation in order to have an idea of the degree of shunt attenuation that can be achieved safely, and often multiple surgeries are needed. Other techniques, like AC or cellophane banding, have been used to produce progressive vein attenuation through chronic ongoing inflammation, but the rate and degree of attenuation has been too variable. Techniques for more controlled progressive attenuation have been developed, involving the use of thrombogenic coils and hydraulic occlusion. Although more expensive and technically more demanding, these techniques are worthy of further investigation.

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REFERENCES

- Barone R. (1976). *Anatomie compare des animaux domestiques*, Vigot, Paris, 665.
- Birchard S.J., Sherding R.G. (1992). Feline portosystemic shunts. *Compendium of Continuing Education of the Practicing Veterinarian* 14, 1295-1300.
- Brooksby GA, Donald DE. (1971). Dynamic changes in splanchnic blood flow and blood volume in dogs during activation of sympathic nerves. *Circulation Research* 29, 227-238.
- Burton C.A., White R.N. (2001). Portovenogram findings in cases of elevated bile acid concentrations following correction of portosystemic shunts. *Journal of Small Animal Practice* 42, 536-540.
- Bussadori R., Bussadori C., Millan L., Costilla S., Altonaga J.J.Asuncion Orden M., Gonzalo-Orden J.M. (2007). Transvenous coil embolisation for the treatment of single congenital portosystemic shunts in six dogs. *The Veterinary Journal, doi.10.1016/j.tvjl.2007.02.027* (article in press)
- De Rycke L., Simoens P., Lauwers H. (1995). Morfologische basis van portosystemische shunts bij de hond. *Vlaams Diergeneeskundig Tijdschrift 64*, 163-172.
- Gonzalo-Orden J.M., Altonaga J.R., Costilla S. (2000). Transvenous coil embolization of an intrahepatic portosystemic shunt in a dog. *Veterinary Radiology and Ultrasound*, 41, 516-518.
- Hottinger H.A., Walshaw R., Hauptman J.G. (1995). Long-term results of complete and partial ligation of congenital portosystemic shunts in dogs. *Veterinary Surgery 24*, 331-336.
- Hunt G.B., Hughes J. (1999). Outcomes after extrahepatic portosystemic shunt ligation in 49 dogs. *Australian Veterinary Journal* 77, 303-307.
- Hunt G.B., Kummeling A., Tisdall P.L.C., Marchevsky A.M., Liptak J.M., Youmans K.R., Goldsmid S.E., Beck J.A. (2004). Outcomes of cellophane banding for

- congenital portosystemic shunts in 106 dogs and 5 cats. *Veterinary Surgery 33*, 25-31.
- Hurn S.D., Edwards G.A. (2003). Perioperative outcomes after three different single extrahepatic portosystemic shunt attenuation techniques in dogs: partial ligation, complete ligation and ameroid constrictor placement. *Australian Veterinary Journal 81*, 666-670.
- Ikeda K., Hayashida Y., Suita S. (1981). Gradual occlusion of the portal branch with hepatic artery ligation for unresectable hepatic tumour in children. *Zeitschrift für Kinderchirurgie 32*, 121-128.
- Johnson C.A., Armstrong P.J., Hauptman J.G. (1987). Congenital portosystemic shunts in dogs: 46 cases (1979-1986). Journal of the American Veterinary Medical Association 191, 1478-1483.
- Komtebedde J., Forsyth S.F., Breznock E.M., and Koblik P.D. (1991). Intrahepatic portosystemic venous anomaly in the dog: perioperative management and complications. *Veterinary Surgery 20*, 37-42.
- Kummeling A., Van Sluijs F.J., Rothuizen J. (2004). Prognostic implications of the degree of shunt narrowing and of the portal vein diameter in dogs with congenital portosystemic shunts. *Veterinary Surgery* 33, 17-24.
- Leveille R., Johnson S.E., Brichard S.J.(2003). Transvenous coil embolisation of portosystemic shunt in dogs. *Veterinary Radiology* 44, 32-36.
- Lawrence D., Bellah J.R., Diaz R. (1992). Results of surgical management of portosystemic shunts in dogs: 20 cases (1985-1990). *Journal of the American Veterinary Medicine Association 201*, 1750-1753.
- Matthews K., Gofton N. (1988). Congenital extrahepatic portosystemic shunt occlusion in the dog: gross observations during surgical correction. *Journal of the American Animal Hospital Association* 24, 387-394.
- Mehl M.L.., Kyles A.E., Hardie E.M., Kass P.H., Adin C.A., Flynn A.K., De Cock H.E., Gregory C.R. (2005). Evaluation of ameroid ring constrictors for treatment for single extrahepatic portosystemic shunts in dogs: 168 cases (1995-2001). Journal of the American Veterinary Medicine Association 226 (12), 2020-2030.
- Murphy S.T., Ellison G.W., Long M., Van Gilder J. (2001). A comparison of the ameroid constrictor versus ligation in the surgical management of single extrahepatic portosystemic shunts. *Journal of the American Animal Hospital Association* 37, 390-396.
- Partington B.P., Partington C.R., Biller D.S. (1993) Transvenous coil embolisation for treatment of patent ductus venosous in a dog. *Journal of the American Veterinary Medicine Association* 202, 281-284.
- Schulz K.S., Martin R.A., Henderson R.A. (1993). Transsplenic portal catheterization: surgical technique and use in two dogs with portosystemic shunts. *Veterinary Surgery* 22, 363-369.
- Seagard J.L., Elegbe E.O., Hopp F.A.(1983). Effects of isoflurane on the baroreceptor reflex. *Anesthesia* 59, 511-520.
- Swalec K.M. and Smeak D.D. (1990). Partial versus complete attenuation of single portosystemic shunts. *Veterinary Surgery* 19, 406-411.
- Swalec K.M., Smeak D.D., Brown J. (1991). Effects of mechanical and pharmacologic manipulations on portal pressure, central venous pressure, and heart rate in dogs. *American Journal of Veterinary Research* 52 (8), 1327-1335.
- Swalec K.M. (1993). Portosystemic shunts. In: Bojrab

- M.J., Bloomberg M.S., Smeak D.D. (editors). *Disease mechanisms in small animal surgery*. 2nd ed., Lea & Febiger, Philadelphia, 298-305.
- Strombeck D.R., Breznock E.M., Mc Neal S. (1977). Surgical treatment for portosystemic shunts in two dogs. *Journal of the American Veterinary Medical Association* 170, 1317-1319.
- Tobias K.M. (2003). Portosystemic shunts and other hepatic vascular anomalies. In: Slatter D. (editor). *Textbook of Small Animal Surgery*. 3rd ed Vol. 1, WB Saunders, Philadelphia, 727-752.
- Tobias K.M.S., Seguin B., Johnston G. (1998). Surgical approaches to single extrahepatic portosystemic shunts. *Compendium on the Continuing Education for the Practicing Veterinarian* 20, 593-601.
- Tobias K.M., Rawlings C.A. (1996). Surgical techniques for extravascular occlusion of intrahepatic shunts. *Compendium of Continued Education of Practicing Veterinarian* 18, 745.
- Van Vechten B.J., Komtebedde J., Koblik P.D. (1994). Use of transcolonic portal scintigraphy to monitor blood

- flow and progressive postoperative attenuation of partially ligated single extrahepatic portosystemic shunts in dogs. *Journal of Veterinary Medicine Association 204*, 1770-1774.
- Winkler J.T., Bohling M.W., Tilson M.D., Wright J.C., Ballagas A.J. (2003). Portosystemic shunts: diagnosis, prognosis, and treatment of 64 cases (1993-2001). *Journal of the American Animal Hospital Association* 39, 169-185.
- Wolschrijn C.F., Mahapokai W., Rothuizen J., Meyer H.P., Van Sluijs F.J. (2000). Gauged attenuation of congenital portosystemic shunts: results in 160 dogs and 15 cats. *Veterinary Quarterly* 22, 94-98.
- Youmans K.R.and Hunt G.B. (1998). Cellophane banding for the gradual attenuation of single extrahepatic portosystemic shunts in eleven dogs. *Australian Veterinary Journal* 76, 531-537.
- Youmans K.R., Hunt G.B.(1999). Experimental evaluation of four methods of progressive venous attenuation in dogs. *Veterinary Surgery* 28, 38-47.